CHAPTER 6

FOOD SAFETY ISSUES AND THE MICROBIOLOGY OF BEEF

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6.1 INTRODUCTION

World demand for high-quality animal protein presents opportunities for growth and expanded trade, which is predicted to increase more than 6% for major beef-producing countries and their beef industries (USDA-FAS, 2006, 2007). Contingent upon increased consumer demand for beef is the production of high-quality, microbiologically safe products. An enhanced stringency of food safety standards has increased the burden for producers and processors to regulate and document their production practices and to implement pathogen control practices. From a food safety standpoint, bacterial pathogens of major concern to beef include enterohemorrhagic *Escherichia coli* (especially *E. coli* O157:H7), *Salmonella*, *Campylobacter*, and *Listeria* (Swartz, 2002). The annual economic loss in 2000 associated with these bacterial pathogens was $5 to 6 billion (Murphy et al., 2003).

6.2 ENTEROHEMORRHAGIC *ESCHERICHIA COLI* O157:H7 IN BEEF

Pathogenic *E. coli* (see Chapter 2) fall into six major categories: enterotoxigenic, enteroinvasive, enteroaggregative, diffusely adherent, enteropathogenic, and enterohemorrhagic (Feng, 2001). Enterohemorrhagic *E. coli* cause hemorrhagic colitis in humans. The disease typically manifests after a 3- to 4-day incubation period as a severe diarrhea that progresses within 3 days to bloody diarrhea in 90% of cases; acute abdominal cramping and vomiting but rarely fever accompany the disease, which lasts about 2 to 9 days (Feng, 2001; Karch et al., 2005). In about 3 to 7% of total cases and about 15% of cases involving children less than 10 years of age, a complication

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of the disease known as hemolytic uremic syndrome (HUS) can result (Feng, 2001; Karch et al., 2005). This syndrome manifests as microangiopathic hemolytic anemia, thrombocytopenia, and intravascular hemolysis and can cause renal failure leading to death in 3 to 5% of cases and to permanent kidney and/or neurological damage in many of the other cases (Feng, 2001; Karch et al., 2005).

Enterohemorrhagic *E. coli* possess a number of virulence attributes, including genes for one or both Shiga toxins (*stx1* and *stx2*), enterohemolysin (*ehxA*), and intestinal adherence factors associated with the locus of enterocyte effacement (LEE), including intimin (*eae*), the translocated intimin receptor (*Tir*), and secreted protein encoded by *EspA, EspB,* and *EspD* (Law, 2000; Nataro and Kaper, 1998). These and, potentially, others traits contribute to the high pathogenicity of this pathogen to humans, as the infectious dose is as low as 10 to 100 cells (Feng, 2001; Karch et al., 2005). Whereas *E. coli* O157:H7 is probably the best known, numerous other EHEC or Shiga toxin–producing serotypes exist (Feng, 2001; Hussein, 2007).

Escherichia coli O157:H7 has been particularly problematic for the beef industry, costing an estimated $2.7 billion loss to the U.S. beef industry alone during the first 10 years since the Jack-in-the-Box outbreak (Kay, 2003). Whereas *E. coli* O157:H7 is estimated to cause a small proportion (0.5% or 62,458 cases) of the total foodborne-caused illnesses in the United States each year (Mead et al., 1999), large outbreaks, with particularly drastic consequences to young children, have attracted media and thus consumer attention to this pathogen. Of the total estimated foodborne-caused hospitalizations, 3% or 1843 are attributed to *E. coli* O157:H7, as are 52 deaths (2.9% of total) (Mead et al., 1999). Large outbreaks associated with the consumption of contaminated ground beef, such as an outbreak affecting 732 people in 1992–1993 in the western United States, of which 55 (mostly children) developed HUS, resulting in the death of four children, have implicated cattle as an important reservoir (Karch et al., 1999). Other ruminants, such as sheep, deer, and goats, can be reservoirs of *E. coli* O157:H7 or Shiga toxin–producing *E. coli*, as can feral and domestic pigs, horses, dogs, seagulls, and house flies (Feng, 2001; Karch et al., 1999, 2005; Naylor et al., 2005). The largest outbreak, due to consumption of radish sprouts served in the school lunch program, occurred in 1996 in Sakai City, Osaka, Japan, and affected more than 8000 people, of which 106 were children, resulting in three deaths (Karch et al., 1999; Michino et al., 1999). Other sources of infections to humans include unpasteurized apple cider or milk, produce, salami, fried potatoes with cheese and spices, potato salad, mayonnaise, yogurt, salmon roe, homemade venison jerky, contact with animals at petting zoos, and contaminated municipal water and swimming pools (Buchanan and Doyle, 1997; Feng, 2001). Interpersonal contact, particularly between family members and attendees of day care centers, has also been documented as a means of *E. coli* O157:H7 transmission (Feng, 2001; Karch et al., 2005).

6.2.1 Prevalence

Human infections peak in summer and early autumn, which coincides with peak fecal shedding by cattle (Bach et al., 2002b; Karch et al., 1999; Naylor et al., 2005;

Rasmussen and Casey, 2001; Renter and Sargeant, 2002); however, considerable variation in prevalence exists between and even within geographic regions. Practically all cattle herds in the United States contain at least some animals colonized by *E. coli* O157:H7, although animal prevalence rates can vary from 0 to >30%, with prevalence being similar in beef and dairy cattle (Bach et al., 2002b; Elder et al., 2000; Rasmussen and Casey, 2001; Renter and Sargeant, 2002). In general, prevalence rates have been found to be higher in the years following the implementation of more sensitive detection methods, such as immunomagnetic separation, than in years before the use of such methods (Gansheroff and O'Brien, 2000; Naylor et al., 2005). More recently, for instance, Khaitsa et al. (2007) reported prevalence as high as 80% in feedlot cattle. In their study, three stages of infection, pre-epidemic, epidemic, and post-epidemic, were observed, and the incidence of shedding was most frequent and the duration of fecal shedding was longest during the epidemic stage.

Prevalence rates in an examination of Finnish cattle were reported to be 1.3% of total cattle tested and ranged from 0 to 6.9%, depending on the abattoir (Lahti et al., 2003). In the United Kingdom, 7.5% of cattle at slaughter yielded *E. coli* O157:H7-positive fecal specimens, and 40% of the farms had at least one animal testing positive for the pathogen (Omisakin et al., 2003). Prevalence rates reported are: Brazil, 1.5%; Japan, 1.8%; Australia, 1.9%; and Scotland, 25% (Naylor et al., 2005). In the Netherlands, prevalence rates from two studies reported that 10.6% of slaughter cattle and from 0.8 to 22.4% of cattle on tested dairy farms were positive for *E. coli* O157 (Heuvelink et al., 1998a, b). *Escherichia coli* O157:H7 was recovered from only one (0.5%) of 200 cattle tested in Argentina, although other Shiga toxin–producing *E. coli* serotypes were isolated from 86 (39%) of these animals (Meichtri et al., 2004). Shiga toxin–producing *E. coli* were isolated on 95% of farms tested between 1993 and 1995 in Spain and from 0 to 100% of the cattle on the farms, with an overall animal prevalence rate of 37% in calves and 27% in cows; however, only 8 (0.7%) of the 1069 cattle tested were positive for *E. coli* O157:H7 (Blanco et al., 2003). From 1993 to 1999 the recovery rate of *E. coli* O157:H7 from 161 calves tested was 0.6%, 0% from 525 cows, 2% from 383 slaughter cattle, and 12% from 471 fed calves, and the authors concluded that these rates were similar to those found elsewhere in Europe and North America (Blanco et al., 2003).

Conedera et al. (2001) reported that *E. coli* O157 was isolated from approximately 4% of 341 dairy calves in one survey and was isolated from 10.7% of a total of 1293 rectal swabs collected from between 92 and 59 animals over an 11- to 15-month period, with peaks as high as 23.7% in summer months. In a Norwegian study, only two of 197 cattle herds had *E. coli* O157:H7-positive fecal specimens (Vold et al., 1998), and *E. coli* O157 was recovered from only 1.25% of 240 (120 dairy and 120 beef) cattle in Mexico (Callaway et al., 2004). Up to 35% of dairy cows shed *E. coli* O157:H7, with nearly twice as many lactating as nonlactating cows shedding *E. coli* O157:H7 (Fitzgerald et al., 2003). Neither parity nor number of days in the milking cycle affected shedding of *E. coli* O157:H7 (Fitzgerald et al., 2003).

6.2.2 Gastrointestinal and Pen Ecology

Most *E. coli* are commensal inhabitants of the gastrointestinal tract and because they are common constituents of excreted feces, often finding their way into water, soil, and sediment (Durso et al., 2004), they have been used extensively as indicators of fecal contamination of food or water (Feng, 2001). Feces, manure, feed, feed bunks, drinking water, and house flies harbor *E. coli* O157:H7 (Alam and Zurek, 2004; Bach et al., 2002b; Duffy, 2003; LeJune et al., 2001; Lynn et al., 1998; Rice and Johnson, 2000), and these sources are thought to play a large role in the dissemination of the organism throughout the herd. In pen environments, exposure and reexposure to these various inoculum sources as well as by animal-to-animal contact probably contribute to the apparently cyclic and transient infection and reinfection of cattle by *E. coli* O157:H7 (Rasmussen and Casey, 2001; Renter and Sargeant, 2002).

In nonfasted cattle, generic *E. coli* are typically present at higher concentrations than *E. coli* O157:H7. For instance, generic *E. coli* were present at about 10^3 to 10^4 CFU/mL in ruminal contents and approximately 10^5 to 10^7 CFU/g in feces (Anderson et al., 2002, 2005; Fegan et al., 2004). By comparison, concentrations of *E. coli* O157:H7 in calves experimentally inoculated with 2×10^{11} CFU did not persist, declining rapidly from an initial high of about 10^4 to 10^5 CFU/mL ruminal fluid 2 h post-inoculation to levels detectable by enrichment only by 3 days post-inoculation (Grauke et al., 2002). *Escherichia coli* O157:H7 concentrations in the feces of these experimentally inoculated calves were first detected 6 h after inoculation and then declined from a high of approximately 10^5 CFU/g achieved 1 day post-inoculation to levels detectable by enrichment only by day 7 post-inoculation (Grauke et al., 2002). Similarly, Buchko et al. (2000) observed that experimentally inoculated *E. coli* O157:H7 populations were rapidly depleted from the rumen of steers but recovered from feces for up to 67 days post-inoculation, thereby indicating that the lower gastrointestinal tract is a more important colonization site than the rumen. In naturally colonized animals, fecal concentrations of *E. coli* O157:H7 in feedlot cattle averaged 1.6×10^3 CFU/g (Cobbold et al., 2007), with fecal specimens containing concentrations higher than that being a rare occurrence (Fegan et al., 2004).

Considerable attention has been directed to the hypothesis that a certain proportion of cattle may shed high numbers of these pathogens (Naylor et al., 2003). It is suspected that even a few of these super-shedding animals within a herd, those shedding more than 10^3 or 10^4 CFU Shiga toxin–producing *E. coli* per gram of feces (depending on the study) may be of greater importance than overall population prevalence per se (Cobbold et al., 2007; Low et al., 2005; Naylor et al., 2003; Omisakin et al., 2003). For instance, Omisakin et al. (2003) reported that while only 9% of 44 infected animals presented to slaughter were found to shed more than 10^4 *E. coli* O157 per gram of feces, these few animals accounted for more than 96% of the total *E. coli* O157 burden shed by all infected animals. Moreover, Ogden et al. (2004) reported that concentrations of *E. coli* O157 in feces of high-shedding animals is greater in the summer than the winter, and this may contribute to the high seasonal rate of human infections. The higher rate of *E. coli* O157 shedding observed

in the summer months has not yet been explained fully, although a recent hypothesis by Edrington et al. (2006a) proposed that hormonal changes associated with longer daylight intervals may be contributing. It is now thought that *E. coli* O157:H7 super-shedders harbor the organisms primarily within a 1- to 15-cm segment of the rectum just proximal to the rectal–anal junction and that this site may be a site of true colonization and attachment (Low et al., 2005; Naylor et al., 2003).

Numerous studies have examined the effect of diet, ionophores, and fasting on fecal *E. coli* O157:H7 shedding, with mixed results (Wells et al., 2009). Diez-Gonzalez et al. (1998) reported that feeding a 90% concentrate diet increased concentrations of generic *E. coli* populations 100-fold compared to concentrations in cattle fed a timothy hay diet. Moreover, the *E. coli* recovered from the concentrate-fed cattle were considerably more resistant to acid shock, purportedly due to increased exposure to higher volatile fatty acid concentrations that resulted from the feeding of more readily fermentable substrates (Diez-Gonzalez et al., 1998). Acid resistance is considered by some to increase the virulence of gut pathogens such as *E. coli* O157:H7 by promoting their ability to survive low-pH, high-gastric acid conditions in the human stomach (Price et al., 2000). Others also found that feeding diets high in forage reduced *E. coli* concentrations or shedding (Callaway et al., 2003b; Gilbert et al., 2005; Gregory et al., 2000; Jordan and McEwen, 1998), but this concept has been challenged. For instance, Hovde et al. (1999) found that experimentally inoculated cattle fed grain or medium- to low-quality hay shed similar concentrations of *E. coli* O157:H7 and that acid resistance of the *E. coli* O157:H7 recovered was unaffected by the diet. Moreover, they reported that the forage-fed cattle shed detectable levels of *E. coli* O157:H7 longer (39 to 42 days) than did grain-fed cattle, which shed the inoculated strain an average of 4 days (Hovde et al., 1999). Van Baale et al. (2004) also observed that cattle fed roughage shed higher numbers of *E. coli* O157:H7 and for longer duration than cattle fed a grain diet. Diets containing barley rather than corn have also been shown to significantly support increased shedding of *E. coli* O157:H7, with one study reporting an increase in prevalence from 38.2% or 50% in steers fed either an 85% cracked corn or 70% : 15% barley/cottonseed diet to 63.2% in steers fed an 85% barley diet (Buchko et al., 2000). In a subsequent study, however, *E. coli* O157:H7 shedding rates in cattle decreased from 2.4% to 1.3%, and concentrations shed decreased only from 3.3 $\log_{10}$ to 3.0 $\log_{10}$ CFU per gram of feces for cattle fed corn or barley finishing diets, respectively (Berg et al., 2004). Thus, the actual impact of such marginal differences on ultimate carcass safety is questionable in the latter study.

Fasting or feed deprivation conditions often associated with transportation of cattle to slaughter have long been considered to promote gut environments more favorable to *E. coli* by reducing concentrations of inhibitory volatile fatty acids produced during fermentation of feedstuffs (Brownlie and Grau, 1967; Grau et al., 1969; Rasmussen et al., 1993; Wolin, 1969). However, results to date have been conflicting, with some studies suggesting that gut *E. coli* concentrations were increased following a fast (Brownlie and Grau, 1967; Grau et al., 1969) and others finding that fasting had no or mixed effects on ruminal or fecal concentrations of *E. coli*, despite having the expected effect on pH and volatile fatty acid concentrations (Anderson et al., 2002; Cray et al., 1998; Harmon et al., 1999). Moreover, Minihan et al. (2003) found no

effect of shipping or lairage on fecal prevalence of *E. coli* O157 in two cohorts of cattle in Ireland, with prevalences of 18, 13, and 12%, respectively in one cohort and 1.7, 1.7, and 0%, respectively, in the other. Additionally, Barham et al. (2002) observed that respective prevalence of *E. coli* O157 in feces and on hides decreased from 9.5% and 18% before shipping to 5.5% and 4.5%, after shipping, suggesting that feed deprivation does not necessarily promote favorable conditions for growth of *E. coli*. In the study by Anderson et al. (2002), fasting did result in decreased VFA concentrations and a neutralization of the pH in the bovine rumen, but total culturable anaerobes were also decreased, implying that while depletion of nutrients available to support growth probably occurred, it affected the total microbial population. Under such conditions it was reasoned that *E. coli* populations would be no more capable than other indigenous anaerobes of competing for limiting nutrients (Anderson et al., 2002). It is reasonable to speculate, however, that upon refeeding, should such an event occur, *E. coli* may propagate more rapidly than populations of slower-growing anaerobes.

Ionophore antibiotics are commonly fed in beef cattle production systems to improve the efficiency of animal production, and because the timing of their implementation coincides approximately with the first occurrence of human *E. coli* O157:H7 infections, their potential effects on *E. coli* O157:H7 prevalence and shedding have been evaluated (Bach et al., 2002a; Callaway et al., 2003a). In vitro, the ionophore monensin had no inhibitory effect on the growth of *E. coli* O157:H7 when applied at concentrations equivelant to levels fed to feedlot cattle (Bach et al., 2002a) or 10-fold higher (Edrington et al., 2003c). These results were not unexpected, however, as ionophores are typically more effective against gram-positive than against gram-negative bacteria. However, Bach et al. (2002a) noted that because of the differential effects of monensin against gram-positive and gram-negative bacteria, they could not discount the possibility that monensin may indirectly open a niche for *E. coli* O157:H7. Numerous other studies, however, have clearly shown that *E. coli* O157:H7 prevalence and shedding were not increased in ruminants fed monensin or other ionophores (lasalocid, laidlomycin propionate, or bambermycin) (Callaway et al., 2003a; Dargatz et al., 1997; Edrington et al., 2003b, 2006b; Garber et al., 1995; Van Baale et al., 2004).

6.3 *SALMONELLA* IN BEEF

Consumption of food and food products derived from meat- and egg-producing animals is believed to be the main source of foodborne salmonellosis in the United States, with an annual cost ranging in the billions (Bryan, 1980, 1981; Frenzen et al., 1999; St. Louis et al., 1988; Todd, 1989). Symptoms of the disease in humans usually occur over 8 to 72 h and include abdominal pain, nausea, and watery diarrhea (D'Aoust, 2001). Enteriditis, Typhimurium, and Typhi are the three main serotypes isolated worldwide (Herikstad et al., 2002). *Salmonella enterica* serotypes Typhimurium and Dublin are considered to be the primary host-adapted serotypes to cattle, with Dublin being the causative biotype for bovine bacteremia (Rabsch et al., 2002). However, other serotypes, such as Enteriditis, which has been thought to be most associated

with chicken eggs, have also been isolated from beef in foodborne outbreaks (Patrick et al., 2004; St. Louis et al., 1988), and more recently, infection by *Salmonella* serovar Newport in people consuming beef has raised concern as to its possible emergence as a prominent foodborne pathogen (Gupta et al., 2003).

6.3.1 Factors That Influence the Spread of *Salmonella*

Foodborne *Salmonella* spp. are generally widespread in agricultural environments. In a recent study of 18 farms from five states, *Salmonella* serovars were recovered from beef, dairy, poultry, and swine farms (Rodriguez et al., 2006). *Salmonella* have also been recovered at different stages during beef slaughter (Stolle, 1981). In addition to the pre- and post-processing facilities, other routes of transmission have been identified, but only a few have been characterized in detail. Within an animal house, airborne routes have been extensively characterized as a potential route for transmission of *Salmonella* in poultry (Holt et al., 1998; Kwon et al., 1999, 2000a). However, outdoor airborne transmission of pathogens is also possible, and depending on proximity can originate from agricultural or municipal sources (Pillai et al., 1996; Pillai and Ricke, 2002). For cattle feedlots it has been suggested that airborne dust is a potential route not only for the transmission of pathogens, but can predispose susceptibility to bacterial and viral infections (MacVean et al., 1986; Wilson et al., 2002). However, Wilson et al. (2002) recovered lower microbial numbers in feedlot dust than those from previous reports from intensively housed farm animals. Animal feed sources of *Salmonella* have been well documented (Maciorowski et al., 2004, 2006b, 2007; Ricke et al., 2005). Animal by-product ingredients have received the most focus as a reservoir for *Salmonella* (Maciorowski et al., 2004), but contamination can occur at any stage of feed processing, including recontamination after thermal processing (Jones and Ricke, 1994; Maciorowski et al., 2006a, 2007; Ricke, 2005). When Bender et al. (1997) fed *Salmonella* artificially contaminated meat-and-bone meal to fistulated dairy cows, *Salmonella* could be recovered from rumen contents, feces, and mesenteric lymph nodes.

Unlike that found with *E. coli*, transportation of cattle has been reported in numerous studies to predispose animals to increased shedding of *Salmonella*. For instance, Corrier et al. (1990) reported that *Salmonella*-prevalence calves shipped from Tennessee to west Texas increased 0 to 1.5% immediately upon arrival at the feedlot and increased further to 8% after 30 days in the feedlot. In cattle shipped to slaughter, respective prevalence levels of *Salmonella* in feces and on hides increased from 18% and 6% before transport to 46% and 89% at the packing plant (Barham et al., 2002). Others have also observed increased prevalence of *Salmonella* on hides following shipment of cattle to slaughter, (Beach et al., 2002; Reicks et al., 2007). Beach et al. (2002) reported that hide contamination by *Salmonella* increased significantly following transportation to slaughter in both adult and feedlot cattle, from 19.8% to 52.2% and 18% to 56%, respectively. They also reported that while fecal *Salmonella* prevalence increased from 1% to 21% in adult cows shipped to slaughter, the prevalence in feedlot cattle was unaffected (3% vs. 5% before and after shipping, respectively). The authors speculated that high-energy diets fed to the feedlot cattle and their higher

Campylobacter colonization status (>60% vs. <8% in adult cattle) may have contributed to the lack of a transportation effect on fecal shedding of *Salmonella* in these cattle.

6.3.2 *Salmonella* and Rumen Ecology

Part of the variability in *Salmonella* occurrence in beef animals lies with the susceptibility of the rumen environment to *Salmonella* survival. It is traditionally believed that the full-fed ruminant animals possess a rumen considered to be hostile to pathogens such as *Salmonella*, due to the high levels of fermentation (Chambers and Lysons, 1979). However, several factors can mitigate this hostility. Feed deprivation can lead to increased numbers of *Salmonella* in cattle (Brownlie and Grau, 1967; Grau et al., 1969), and in poultry, removal of feed has led to a gut environment much more conducive to expression of virulence genes and subsequent invasion of internal organs (Dunkley et al., 2007; Durant et al., 1999a). Volatile fatty acids (VFAs) are considered to be inhibitory to *Salmonella* growth, but this inhibition is dependent on concentration and degree of acidity (Cherrington et al., 1991; Goepfert and Hicks, 1969; McHan and Shotts, 1993). However, induction of acid tolerance can provide protection against organic acids (Baik et al., 1996) and influence virulence response (Durant et al., 1999b, 2000a–c; Lawhon et al., 2002). Exposure to VFAs at neutral pH can induce resistance to inorganic acids as well as high osmolarity and reactive oxygen (Greenacre et al., 2003; Kwon and Ricke, 1998; Kwon et al., 2000b). Several biological agents exist in the rumen that can directly or indirectly lyse or destroy bacteria, including bacteriophages, bacteriocins, and protozoans. Although anaerobic protozoans typically prey on rumen bacteria, using them as a nutrient source, it has recently been shown that *Salmonella* can survive in these protozoans, and these survivors are more invasive in tissue culture, resulting in *Salmonella* exhibiting a hyperinvasive phenotype (Carlson et al., 2007; Rasmussen et al., 2005).

6.4 *LISTERIA* IN BEEF

The annual economic loss in 2000 associated with foodborne *Listeria monocytogenes* was estimated at $2.3 billion (wwww.ers.usda.gov). During the period October 1, 1993 to September 30, 1998, microbial contamination of food and cosmetic products was the leading cause for recalls, accounting for a total of 1370 recalls (36% of all products recalled). *Listeria monocytogenes* accounted for the greatest number of food products recalled. Nearly two-thirds of all product recalls due to *L. monocytogenes* contamination were dairy products, pastries, salads, or sandwiches (Wong et al., 2000).

6.4.1 Ecology of *Listeria*

Ruminants are often fed forage that is contaminated with *L. monocytogenes* and frequently shed this organism in their feces. Zundel and Bernard (2006) reported that in *Listeria*-free sheep that had been inoculated with *L. monocytogenes*, this pathogen

spread throughout the entire volume of the forestomachs within 4 h and through the entire gastrointestinal tract within 24 h. These sheep shed *L. monocytogenes* for 10 days. *Listeria* persisted for at least 14 days in rumen digest and retropharyngeal lymph nodes and at relatively high levels of about 10^4 CFU/g in palatine tonsils. They concluded that *L. monocytogenes* translocates throughout the digestive tract of asymptomatic sheep, with the exception of the gallbladder, and that brief and low-level fecal excretion of *L. monocytogenes* is concomitant with transitory asymptomatic infection in sheep.

Fenlon (1985) reported that silage containing low levels of oxygen was contaminated with *L. monocytogenes*, whereas silage kept under strict anaerobic conditions with a consistently low pH did not include any *Listeria*. In silage, the strictly anaerobic conditions coupled with the predominance of lactic acid bacteria that reduce the pH results in conditions that are unfavorable for *L. monocytogenes* growth. Damaged silage bags with high amounts of oxygen also did not support *L. monocytogenes* growth, and *L. monocytogenes* was probably outcompeted by aerobic microorganisms. However, the conditions in the silage bales that contained low amounts of oxygen restricted aerobic species, and limited acid production by the lactics allowed the proliferation of *L. monocytogenes*. Therefore, farmers feeding silage to their animals need to take into account the atmospheric status of their silage, as this could be a source of *L. monocytogenes* for susceptible and asymptomatic animals. Microaerophilic conditions in silage may allow the persistence and further dissemination of *L. monocytogenes* in the farm environment.

In addition to the persistence of *L. monocytogenes* observed in bovine manure-amended soil, Nightingale et al. (2004) showed that the bovine farm ecosystem maintains a high prevalence of *L. monocytogenes*, including subtypes linked to human listeriosis cases and outbreaks. It also appears that cattle contribute to amplification and dispersal of *L. monocytogenes* into the farm environment.

6.4.2 Dissemination Factors of *Listeria*

The prevalence of *L. monocytogenes* in bovine and other farm ecosystems presents a challenge to the food industry, where zero tolerance of *L. monocytogenes* on RTE foods is mandated. Not only could beef processing plants be contaminated with *L. monocytogenes* from raw bovine products, but some of these *L. monocytogenes* may persist within the plant environment and thus recontaminate processed RTE beef products.

Control of *L. monocytogenes* in preharvest environments remains elusive. This is due partially to the persistence of the organism in the environment. In a study conducted by Dowe et al. (1997), soil type apparently influenced the survival of *L. monocytogenes*, with sandy soil having the worst long-term prospects for survival. Soils with greater absorption of moisture showed marked *L. monocytogenes* growth. Therefore, moisture levels may also be the most influential abiotic factor in determining *L. monocytogenes* levels. *L. monocytogenes* increased from low inoculum levels but decreased from high inoculum levels and also reached higher levels more rapidly in autoclaved soil. Multiplication of *L. monocytogenes* in these soils strengthens the hypothesis that this environment is a key reservoir for the organism. Interestingly,

this pathogen thrives in the presence of some natural background flora. The presence of reduced microbial competitors in soil amended with solid chicken manure also supported higher populations of *L. monocytogenes* than did soils amended with either liquid hog manure or inorganic nitrogen–phosphorus–potassium fertilizer. It appears that low levels of *L. monocytogenes* such as those shed in fecal matter may provide adequate inoculum to establish a population of *L. monocytogenes* in soil.

In conclusion, *L. monocytogenes* routes of contamination both pre- and post-harvest are better understood, but developing effective control measures for all potential sites of contamination remains difficult. Future work is needed to develop more understanding of this organism when present in low-oxygen and anaerobic environments and how this may influence growth, survival, and pathogenesis.

6.5 *CAMPYLOBACTER* IN BEEF

Campylobacter spp. are now estimated to be the leading bacterial cause of food-borne illness in several developed countries. In the United States it causes 1,963,141 illnesses, 10,539 hospitalizations, and 99 deaths annually (Mead et al., 1999) at an estimated cost of $1,215,300,000 annually (USDA-ERS, 2008). After a 1- to 7-day incubation period, campylobacteriosis involves symptoms such as abdominal cramps, mild to severe inflammatory diarrhea, and bloody stools, which typically last for 2 to 3 days (Ketley, 1997). Campylobacteria can also infrequently cause post-infection complications associated with acquiring immune-mediated neuropathies—Guillain–Barré syndrome or Miller–Fisher syndrome (Jacobs et al., 1998; Nachamkin et al., 1998; Rees et al., 1995; Salloway et al., 1996)—and may potentially contribute to the development of inflammatory bowel diseases such as Crohn's disease (Lamhonwah et al., 2005).

Campylobacter are small, curved-to-spiral-shaped, flagellated gram-negative rods ranging from 0.5 to 8 μm in length and 0.2 to 0.5 μm wide (Penner, 1988). The genus *Campylobacter* is made up of 17 species (Foster et al., 2004; On, 2001); however, in the United States, about 99% of *Campylobacter* infections are caused by *C. jejuni* (CDC, 2005). *Campylobacter coli* is recognized as the next most prevalent food-poisoning species and is estimated to have been responsible for approximately 26,000 cases of intestinal inflammatory responses in 2000 (Gillespie et al., 2002; Tam et al., 2003). These *Campylobacter* appear well adapted to survive and colonize within the digestive tracts of warm-blooded hosts, and while conditions that include a microaerobic atmosphere and temperatures ranging between 37 and 42°C are optimal for growth (Altekruse et al., 1999), *Campylobacter* are capable of surviving on countertops for several days, and transmission to food during preparation in kitchens has been reported (Luber et al., 2006).

6.5.1 Prevalence

Campylobacter jejuni and *C. coli* are natural colonizers of the gastrointestinal tracts of domestic and feral animals and are generally asymptomatic in food production

animals (Stanley and Jones, 2003). Despite early reports of their isolation from cattle (Garcia et al., 1985; Manser and Dalziel, 1985; Munroe et al., 1983), *Campylobacter* have been recognized primarily as important foodborne pathogens in poultry and unpasteurized dairy products (Butzler and Oosterom, 1991). For instance, *C. jejuni* has been recovered at isolation rates as high as 98% from retail poultry products (Altekruse et al., 1999) and 12.3% from bulk tank milk samples (Oliver et al., 2005). Nevertheless, *Campylobacter* are known to be present on dairy farms, with prevalence being higher in lactating cows (42.9%) than in cull cows (30.3%) (Wesley et al., 2000). A recent study reported that prevalence was higher in calves than in cows and higher on smaller than on larger farms in Wisconsin (Sato et al., 2004). This study also reported that prevalence rates were similar (29.1% and 26.7%, respectively) on the conventional and antimicrobial-free dairy farms studied (Sato et al., 2004).

With respect to beef cattle, Garcia et al. (1985) found *C. jejuni* to present more often in steers (55%) than in cows (22%) or bulls and heifers (each at 40%). Conversley, Bae et al. (2005) reported a higher prevalence rate of *C. jejuni* in cow–calf operations (47.1%) than in calf rearing, in a feedlot operation (23.8% and 31.6%, respectively), or in dairy operations (31.2%). Length of time within a feedlot appears to affect colonization status as prevalence of *C. jejuni* in fed cattle increased during feeding from 1.6% to as high as 63% near the finishing period (Besser et al., 2005). Prevalence rates in slaughter cattle, as determined via culture of rectal swabs collected before and after transit, were similar in feedlot cattle (64% to 68%, respectively) and adult cattle (6% to 7%, respectively), thus indicating that transportation had little effect on colonization status (Beach et al., 2002). Hide contamination as determined via a culture of swabs taken at the animals' hindquarter region, decreased during transit from 25% to 13% *Campylobacter*-positive samples in the feedlot cattle but were similar for the adult cattle (1% to 2%, respectfully) (Beach et al., 2002). In cattle, prevalence rates in general have been higher for *C. jejuni* than for *C. coli* (Bae et al., 2005; Harvey et al., 2005; Inglis and Kalischuk, 2003; Inglis et al., 2004), although Bae et al. (2005) found that *C. coli* prevalence was nearly equivalent to that of *C. jejuni* (20% vs. 23.8%, respectfully) in calf-rearing operations. *Campylobacter* prevalence in feedlot cattle has been found in at least one study to be much higher than that of *Salmonella* (Beach et al., 2002). Studies elsewhere have reported *Campylobacter* prevalences in beef cattle to be 24.8% in Northern Ireland (Madden et al., 2007), 53.9% in northeastern Italy (Pezzotti et al., 2003), 31.1% in Finland (Hakkinen et al., 2007), 26% in southwestern Norway (Johnsen et al., 2006), 10.2% in Switzerland (Al-Saigh et al., 2004), and 58% for feedlot cattle and 2% for pasture cattle in Australia (Bailey et al., 2003). Unlike that observed with dairy cattle, beef cattle do not appear to exhibit increased *Campylobacter*-colonization status during the summer months (Stanley et al., 1998).

6.5.2 Gastrointestinal Ecology

Garcia et al. (1985) sampled multiple internal viscera for *C. jejuni* and *C. coli* and successfully recovered *C. jejuni* serotypes from the gallbladder, large intestine, small intestine, liver, and lymph nodes. The gallbladder mucosal tissue and bile have been

found to be good sites for *Campylobacter* colonization (Garcia et al., 1985; Saito et al., 2005) and *Campybacter*-positive liver samples have been recovered from 12% of beef cows sampled and 54.2% of Japanese oxen sampled, with most isolates identified as *C. jejuni* (Kramer et al., 2000). In one study, *Campylobacter* were readily recovered from fecal specimens of feedlot steers but not from ruminal contents of the same animals (Gutierrez-Bañuelos et al., 2007). *Campylobacter jejuni* and *C. coli* are generally asymptomatic in most colonized cattle; however, cases of diarrhea and gastroenteritis in calves have been reported, and this may be one rational for increased antibiotic use within farms and feedlots (Stanley and Jones, 2003).

6.6 CONTROL OF FOODBORNE PATHOGENS IN BEEF

A number of technologies have been developed to reduce contamination of carcasses by foodborne pathogens during slaughter and processing (Castell-Perez and Moreira, 2004; Keeton and Eddy, 2004). The meat industry has generally adopted a multiple-hurdle approach encompassing the training of food handlers in effective hygiene and implementation of postharvest interventions such as hot water and organic acid rinses, steam pasteurization, chemical dehairing, steam vaccuming, and irradiation (Acuff et al., 1987; Belk, 2001; Cherrington et al., 1991; Dickson, 1992; Dorsa, 1997; Farkas, 1998; Hardin et al., 1995; Koohmaraie et al., 2005; Micheals et al., 2004; Ricke, 2003; Ricke et al., 2005). Interventions such as these are intended to minimize contamination of meat products by foodborne pathogens. For instance, despite its ubiquitous dissemination in animals, *Listeria* is considered primarily a food safety risk post-harvest, and subsequently, a wide variety of chemical and physical interventions have been examined and/or proposed (Tompkin, 2002). More recently, Dimitrijevic et al. (2006) demonstrated that several nitro-based compounds decreased growth rates of *L. monocytogenes* during anaerobic culture and aerobic 4°C storage over 4 months.

In the red meat industry, hide removal and evisceration are particularly important critical control points, as these processes have been proposed as most likely to result in the contamination of carcasses (Pearce et al., 2004; Ryan, 2007; Tergney and Bolton, 2006). For beef processors in the United States, the efficacy of post-harvest interventions must be extremely high since *E. coli* O157:H7 is classified as an adulterant by the Food and Drug Administration, which applies a zero tolerance for the pathogen in ground meat (USDA-FSIS, 2004). However, despite Herculean efforts by packers and processors, current post-harvest interventions are not infallible, as product recalls and outbreaks of human foodborne disease continue to occur. In a risk assessment conducted by Cassin et al. (1998), the concentration of *E. coli* O157:H7 in feces of animals at slaughter was the greatest risk factor associated with *E. coli* O157:H7 foodborne illness from the consumption of hamburgers, suggesting that reducing carriage within animals pre-harvest may be beneficial. Moreover, other risk assessments have indicated that pre-harvest interventions would reduce human exposure to pathogens (Hynes and Wachsmuth, 2000; Vugia et al., 2003). Consequently, considerable research has been directed toward the development of interventions that

can reduce the incidence and concentrations of foodborne pathogens in food animals during on-farm rearing; however, minimizing the spread of foodborne pathogens via on-farm measures remains elusive.

On-farm food safety undoubtedly begins with good animal husbandry and farm management, including effective sanitation practices (Collins and Wall, 2004; OIE, 2006). Contaminated feed and poor-quality silages have long been recognized as a potential source of pathogens to livestock operations, with many of the pathogens surviving for several months in dry feeds (Crump et al., 2002; Davis et al., 2003; Fenlon and Wilson, 2000; Lynn et al., 1998; Nightingale et al., 2004; Wilkinson, 1999). Consequently, considerable focus has been directed toward eliminating these sources of infection, particularly *Salmonella,* in animal and poultry feeds (Ha et al., 1998a, b; Juven et al., 1984). The addition of organic acids to repress *Salmonella* in feeds has been the primary set of antimicrobial compounds examined particularly for poultry feed (Hinton and Linton, 1988; Khan and Katamay, 1969; Maciorowski et al., 2004, 2006a).

Once a foodborne pathogen has been ingested by the animal, however, it becomes more difficult to minimize and/or eliminate these pathogens from a complex ecosystem such as the rumen or lower gastrointestinal area without disruption of more beneficial microflora. Antibiotics can be effective as feed supplements, such as has been shown with the use of neomycin to reduce bovine carriage of *E. coli* O157:H7 (Elder et al., 2003; Loneragan and Brashears, 2005), but uncontrolled use may promote the emergence of resistant foodborne pathogen strains of risk to human therapies (Cox et al., 2007).

Considerable research aimed at developing safe chemical feed or water supplements to reduce the incidence, survivability, and virulence of microbial pathogens in the gut of food animals during all stages of production is under way. For instance, the use of an experimental chlorate product to specifically target respiratory nitrate reductase enzymes possessed by *E. coli* and *Salmonella* has recently been investigated. It was hypothesized that an experimental product containing chlorate (ECP) may selectively kill nitrate-respiring *Salmonella* and *E. coli*, which also reduce chlorate to cytotoxic chlorite (Pichinoty and Piéchaud, 1968; Stewart, 1988) without harming beneficial gut bacteria (Anderson et al., 2000). In support of this hypothesis, *Salmonella* serovar Typhimurium DT104 and *E. coli* O157:H7, but not total culturable anaerobes, were reduced more than 10,000-fold during in vitro incubation of buffered ruminal fluid supplemented with 1.25 and 5 mM active chlorate ion (Anderson et al., 2000). Several studies have since demonstrated that intraruminal, drinking water, or feed administration of ECP significantly reduced fecal *E. coli* concentrations (Anderson et al., 2002, 2005; Callaway et al., 2002; Fox et al., 2005). Evidence from these studies indicated that an experimental chlorate product designed to bypass the rumen so as to enhance delivery of the active ion to the lower gut increased bactericidal efficacy in the lower gut (Anderson et al., 2005; Edrington et al., 2003a; Fox et al., 2005). Whereas studies testing ECP against *Salmonella* in cattle have yet to be done, numerous studies have shown significant reductions in *Salmonella* colonization in the alimentary tract of broilers, turkeys, and pigs (Anderson et al., 2001a, b; 2004; Byrd et al., 2003; Moore et al., 2006; Patchanee et al., 2007).

Another potential supplemental feeding strategy involves the administration of select nitroalkanes (i.e., 2-nitropropanol, 2-nitroethane, and 2-nitroethanol) that have been shown to exhibit inhibitory activity against *E. coli* O157:H7, *Listeria*, *Campylobacter*, and *Yersinia* in vitro (Anderson et al., 2007; Horrocks et al., 2007; Jung et al., 2004a). Moreover, the nitroalkanes were shown to reduce *Salmonella* colonization effectively in the gut of broilers (Jung et al., 2004b), and *Salmonella* and *Campylobacter* colonization in pigs (Jung et al., 2003), and to synergistically enhance the bactericidal activity of chlorate against *Salmonella* Typhimurium (Anderson et al., 2006c, 2007). Their efficacy has not yet been demonstrated in cattle (Gutierrez-Bañuelos et al., 2007). An attractive aspect of the nitroalkanes is that these compounds have been shown to be potent inhibitors of enteric methanogenesis (Anderson et al., 2006a; Gutierrez-Bañuelos et al., 2007) as well as against *Listeria* spp. (Dimitrijevic et al., 2006). Thus, the potential could be used to reduce economic and environmental costs associated with ruminal methane production and *Listeria* spp. should the latter be recognized as a preharvest problem. Similarly, the medium-chain fatty acid laurate and its glycerol monoester, monolaurin, also inhibit ruminal methanogenesis and *Listeria* (Božic et al., 2007a, b). The bactericidal effects of laurate and monolaurin probably result from a disruption of the cell wall of gram-positive or gram-positive-type organisms, which includes many ruminal bacteria that contribute to digestion, and thus their use as feed additives throughout the feeding. Additionally, their assimilation into intramuscular or subcutaneous fat may be undesirable from a human health perspective. However, it is not unreasonable to suspect that their use during the last day or several days before slaughter may significantly reduce gut carriage of *Listeria* with minimal effects on production efficiency or fat accretion. Another preharvest food safety strategy that captures economic benefits for livestock producers is the commercial dietary supplement, Tasco-14 (a preparation of the marine seaweed *Ascophyllum nodosum*), which has positive effects on carcass quality and product shelf life (Braden et al., 2007). When fed to feedlot cattle at 2% of the diet dry matter, Tasco-14 reduced incidence of *E. coli* O157-positive on hide swabs by more than 30% and feces by more than 9% (Braden et al., 2004). Fecal samples from the Tasco-14 supplement cattle also had less *Salmonella* than did nonsupplement cattle at the end of the feeding period (Braden et al., 2004).

Biocontrol methods employing the use of lytic bacteriophages are presently receiving much research emphasis as potential strategies to reduce the carriage of foodborne pathogens, being spurred on by the recent approval of an anti-*Listeria* phage spray for processed meat and poultry (Joerger, 2003; Strauch et al., 2007). Kudva et al. (1999) reported anti-*E. coli* O157:H7 lysis by specific bacteriophages and application of lytic bacteriophages to the rectoanal junction of experimentally inoculated cattle significantly lowered concentrations of *E. coli* O157:H7 recovered from this site (Sheng et al., 2006). Raya et al. (2006) reported 2-log-unit reductions in *E. coli* O157:H7 in sheep by 2 days post-administration. Lysis by bacteriophages specific for *Salmonella* and *Campylobacter* has been attempted with mixed success in poultry. In broliers, Wagenaar et al. (2005) reported 3-log reductions of *C. jejuni* by 3 days post-phage administrion, and Loc Carrillo et al. (2005) reported 0.5- to

5-log reductions of *C. jejuni* within 5 days of treatment. Phage therapy to broilers has been shown to reduce colinization by the *Salmonella* serovar Enteritidis by 0.3 to 3.5 log units (Fiorentin et al., 2005; Sklar and Joerger, 2001).

Preventing initial establishment and colonization of *Salmonella* in the animal would appear to be the optimal approach. Generation of antibodies either as a feed amendment or via a genetically engineered plant or grain that can be fed has some merit but may be cost prohibitive (Berghman et al., 2005). Beneficial probiotic and competitive cultures, the latter named for their purported ability to exclude by outcompeting the pathogen, have been used successfully in poultry to limit colonization in the gut (Anderson et al., 2006b). These approaches, however, have typically involved young birds with a minimal microflora present prior to introduction of the probiotic (Nisbet et al., 1994, 1996a, b), and thus this type of intervention in theory might prove to be more difficult to establish in the more complex ruminant ecosystem, where functionality of competitiveness is less well understood (Ricke and Pillai, 1999). Nevertheless, beneficial effects of administering probiotic lactic acid or nonpathogenic colicin-producing *E. coli* bacteria on reducing the incidence of shedding of *E. coli* O157:H7 in cattle and on hides have been reported (Brashears et al., 2003a, b; Elam et al., 2003; Schamberger et al., 2004; Younts-Dahl et al., 2004; Zhoa et al., 1998, 2003).

Immunizing young animals such as calves offers the opportunity to use the animal's immune system to ward off future systemic infections after exposure to foodborne pathogens later in life (Mastroeni et al., 2000). Parenteral vaccinations of young calves against *S.* Typhimurium using an auxotrophic-attenuated live strain limited the clinical signs expressed in calves exposed to the virulent version of the strain (van der Walt et al., 2001). Vaccination of cattle with components of the type III secretory system has been shown to help reduce shedding of *E. coli* O157:H7 in cattle. Potter et al. (2004) reported that vaccination reduced both the incidence (15 vs. 57 incidents of shedding out of 112 possible incidents over 14 days by vaccinated or nonvaccinated cattle, respectively; $n = 8$ per group) and concentration of *E. coli* O157:H7 shedding (6.25 vs. 81.25 CFU/g of feces for vaccinated and nonvaccinated cattle, respectively). In a subsequent study, however, vaccination with the type III immunogens was ineffective in reducing prevalence of *E. coli* O157:H7 (Van Donkersgoed et al., 2005). Thus, it is clear that a more in-depth understanding of the factors that influence virulence response of foodborne *Salmonella* and enterohemorrhagic *E. coli* in beef cattle is needed. Given the broad host range and multiple serotypes of foodborne pathogens, the development of multivalent vaccines against *Salmonella* and possibly against enterohemorhaggic *E. coli* may be needed to achieve better effectiveness (Wallis, 2001). In the case of *Salmonella*, for instance, pathogenesis requires multiple genes for complete virulence expression and can be regulated by a number of environmental factors, including anaerobiosis and VFA (Durant et al., 2000b; Ernst et al., 1990; Francis et al., 1992; Lucas and Lee, 2000; Marcus et al., 2000; Singh et al., 2000). Complete sequencing of foodborne pathogens coupled with implementation of newer molecular screening tools such as transposon footprinting and microarray analysis should further delineate virulence responses (De Keersmaecker et al., 2005; Hayashi et al., 2001; Kwon and Ricke, 2000, Kwon et al., 2002; Lucchini et al., 2001; Marchal

et al., 2004; McClelland et al., 2001; Parkhill et al., 2000) and enable the construction of optimal genetic vaccine constructs.

Effective control of foodborne pathogens will also potentially rely on sensitive and rapid detection during the early states of their establishment in the beef environment. A myriad of cultural, immunological, and molecular methods have been employed for detection and identification of pathogens in various environments and sample matrices (see Chapter 27) (Bettelheim and Beutin, 2003; Gasanov et al., 2005; Gracias and McKillip, 2004; Kulkarni et al., 2002; Maciorowski et al., 2006b; Petrenko and Sorokulova, 2004; Ricke, 2005). Molecular detection using polymerase chain reaction approaches have been successful but are limited by their inability to distinguish nonviable from viable cells in feed (Maciorowski et al., 2000, 2005). Newer approaches that involve direct measurement of gene expression would resolve some of these issues. To illustrate, application of microarray technology provides an opportunity to screen rapidly for specific strains of *Salmonella* (Goldschmidt, 2006; Maciorowski et al., 2005; Nutt et al., 2004). However, standardization of these as well as conventional cultural methodologies between laboratories remains a problem (Gracias and McKillip, 2004; Malorny et al., 2003).

6.7 CONCLUSIONS

Enterohemorrhagic *E. coli, Salmonella, Listeria*, and *Campylobacter* remain foodborne pathogens of significance to the beef industry. The annual economic loss in 2000 associated with these foodborne pathogens was estimated at $5 to 6 billion (Murphy et al., 2003). Considerable research has yielded important information pertaining to the epidemiology and ecology of these pathogens in cattle, and progress has been made toward the development of interventions to minimize their carriage in animals. Preharvest interventions such as the seaweed preparation, Tasco-14, and probiotic mixtures of lactic acid bacteria are Generally Recognized as Safe (GRAS) within the United States and with such status they are commercially available. An anti-*E. coli* O157:H7 vaccine for cattle has been approved by the Canadian Food Inspection Agency for use in Canada. Interventions employing chlorate or nitrocompounds await regulatory approval from agencies such as the U.S. Food and Drug Administration. Challenges remain for the beef industry; however, as issues that extend well beyond the pathogens discussed in this chapter, including the emergence of existing and new pathogens, the emergence and spread of antimicrobial-resistant bacteria and environmental issues come to the forefront.

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